Research Paper

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Study of Homocysteine, High Sensitive Troponin-I and CK-MB in Myocardial Infarction.

Clinical Research

KEYWORDS : Myocardial infarction, High Sensitive Troponin I, Cardiac biomarkers, Homocysteine.

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ABSTRACT

Myocardial infarction (MI) is a major consequence of coronary artery disease .Apart from the traditional risk factors , other novel risk factors are also found to play a major role in the progression of myocardial infarction. Of the novel risk factors, hyperhomocysteinuria is the most important culprit in the development of MI. Homocysteine is an independent and graded risk factor for vascular thrombosis .It is involved in the progression of atherosclerosis, stroke, and peripheral thrombosis. Hyperhomocysteinuria is a treatable hereditary disorder and if detected sufficiently early can modify or delay the onset of vascular diseases .In the present study cardio biomarkers, high sensitive troponin I, CK-MB, and homocysteine levels are measured in MI patients and compared with healthy controls .As expected, high sensitive troponin I and CK-MB are elevated in male and female MI patients. Homocysteine is definitely an independent risk factor and it is found to be elevated in 80%of male and 50% of female MI patients. Hence assessing its levels at an early stage and controlling the levels, could prevent or reduce the onset of MI in hyperhomocysteinemia. Hence it is the often neglected risk factor which is the culprit not only for MI but also for stroke, peripheral sclerosis and other types of thrombosis.

INTRODUCTION

Several studies have been carried out on the role of risk factors in the development and onset of atherosclerosis leading to acute MI. These studies are aimed at lowering the rate of cardiovascular diseases especially atherosclerosis since this is the major cause of mortality and morbidity in south Asia. High incidence of coronary heart diseases is reported in India and the reported cases number 1.17millions in 1970 and 2.013 millions in 2010(1).

Genetic factors and altered life style are alleged to be the major causative agents leading to cardiac diseases, the former being non-modifiable and the latter being definitely modifiable. Advancing age,dislipidemia,family history of MI at an early age ,diabetes mellitus and high systolic blood pressure are the classical risk factors associated with the first group .Constant and continued search for other than the accepted risk factors have brought to light the significant role of lipo(a),fibrinogen and homocysteine leading to the development of atherosclerosis .

Coronary heart disease in Indians is often seen at middle ages and follows a malignant course of progression .It is observed that the incidence of stroke in youngsters below the age of 45 years is increasing year after year in India .So much so, cardiovascular diseases and stroke are the major contributors to mortality and disability in south Asia ,including India (2).

Homocysteine is now accepted as an independent graded risk factor for cardiao vascular diseases (3).Indian population and those migrated to USA or UK have 6% higher fasting homocysteine levels compared to Europeans and North Americans (4). Hyperhomocysteineuria may be attributed to the incidence of twice as many coronary heart diseases in Indian Asians as compared to their US counterparts (5).Homocysteine levels are higher in males than in females and it increase with age (6).Moreover ,vegetarians possess higher homocysteine levels probability due to low levels of Vitamin (7).

In the present study, levels of cardiosensitive and specific biomarkers and also homocysteine are measured in controls and MI patients with a view to understand any possible role for homocysteine in the progression of atherosclerosis.

MATERIALS AND METHODS

Patients admitted to SK Hospital IP form the core of this study .The patients have been diagnosed by expert cardiologists based on patient history ,levels of biomarkers and ECG changes .30 male patients and 20 female patients are included in the present study.

METHODS:

High sensitive Troponin I ,CK-MB and Homocysteine are measured by chemiluminescence microparticle immunoassay using ARCHITECT i1000SR of ABBOTT.

RESULTS

Table I depicts the levels of highsensitive troponin I, CK-MB and homocystiene in male MI patients and controls .Though there is an increase in high sensitive troponin, levels,because of the wide variation in its values ,this increase is not statistically significant .In male MI patients CK-MB levels are elevated significantly as compared to controls .This elevation is statically significant (<0.001).

Homocysteine levels are higher in 80% of male MI patients as compared to controls. In the remaining 20% of MI patients homocysteine levels are near normal.

Levels of serum high sensitive troponin I , CK-MB and homocysteine are measured in female MI patients and compared with controls .Evaluation of high sensitive troponin I is significant statistically (<0.01) as compared to controls. The increase in CK-MB is also statistically highly significant (<0.001) in MI female patients as compared to controls .Homocysteine levels increased in half the number of female patients (50%) while in the other half the levels are almost normal.(Table No. 2)

DISCUSSION

Extensive and exhaustive studies by several authors attempting to reduce the incidence of MI by evaluating the classical genetic and modifiable risk factors as well as to look into newer novel risk factors have come to conclude that Lipo(a),fibrinogen and homocysteine are associated with acute MI and are also useful novel risk factors(8).

High sensitive troponin I and CK-MB are elevated in MI patients

as compared to controls. This increase is in agreement with several other reports (9).

The association of homocysteine and atherosclerosis is first suggested by McCully in 1969(10).It is now well established that the elevated plasma homocysteine is a strong, graded independent risk factor for MI, stroke and other vascular diseases (11). Homocysteine levels varying between 5-15 µmol/L, 16-30 µmol/L and >100 µmol/L are classified into three groups viz mild, moderate and severe homocysteineuria , respectively(12).Vitamin ,folic acid and pyridoxine are the vitamins involved in methionine metabolism. Homocysteine is produced during this metabolism .Deficiency of all these or any one of them can lead to hyperhomocysteinuria. A rise in homocysteine above 90^{th} or 95^{th} percentile of control is accompanied by increased risk of fatal and nonfatal atherosclerotic artery diseases.

Homocysteinuria is a treatable hereditary disorder and if untreated may lead to coronary artery disease, peripheral artery diseases and various other types of thrombosis (13).Homocysteine may induce atherosclerosis by affecting endothelial derived relaxing factor nitric oxide(NO). Nitric oxide combines with homocyteine in presence of Oxygen (O_2) to form S-nitroso-L-homocysteine which inhibits production of H_2O_2.The bioavailability of NO is reduced leading to endothelial injury .This could be due to production of free radicals by homocysteine. Moreover , homocysteine enhances lipid peroxidation which could decrease the production of NO(14).Autooxidation of homocysteine results in oxidation of LDL through formation of superoxide anion . Homocysteine could lower antioxidant status which could damage endothelial cells.Homocysteine stimulate platelets aggregation of thromboxane A_2 which is a vasodialator and proaggregant (8).Homocysteine may interact with cholesterol by increasing LDL oxidation thus predisposing to atherosclerosis. Homocystiene is an independent risk factor but all acute MI patients do not show elevated homocysteine levels.Correlation between MI and hyperhomocysteinmia is observed in 80% of male and 50% of female patients. Hence homocysteine could be an independent risk factor, which when managed clinically ,could substantially lower MI.

CONCLUSIONS

Homocysteine is an often neglected but common culprit of coronary heart diseases .Thrombosis rather than atherosclerosis is the main culprit for vascular complications associated with high homocysteine levels.

Hyperhomocysteinemia is a treatable genetic disorder but can also occur in Vitamin B_{12}, folic acid and pyridoxine deficiency especially in vegetarians. If detected early it can be cured by vitamin supplementation or by altering concentration of dietary methionine .It is a neglected risk factor for vascular diseases .Detection of homocysteinemia at an early stage and controlling it could reduce the incidence of thrombosis .Curtailing the incidences of atherosclerosis and stroke could considerably reduce the expenses of health care professionals and Governments. Strategies like public awareness, measurement of homocysteine as a compulsory screening test from birth and its proper management in all vascular diseases including atherosclerosis will be a useful step with far reaching benefits in health care management.

Table No. 1: Levels of High Sensitive Troponin I ,CK-MB and Homocysteine in Male MI Patients and Controls.

		NUMBER	MEAN±SD	P value
High Sensitive Troponin-I	Case	30	2327.81±8351.72	0.11
	Control	30	9.11±3.1	
CK-MB	Case	30	5.61±4.68	0.0001
	Control	30	1.57±0.92	
HOMOCYSTEINE (Positive-Cases)	Case	24(80%)	15.32±4.98	0.0001
	Control	24	9.11±3.1	
HOMOCYSTEINE (Negative Cases)	Case	6(20%)	7.20±2.47	0.12
	Control	6	9.11±3.1	

TableNo. 2: Levels of High Sensitive Troponin I ,CK-MB and Homocysteine in Female MI Patients and Controls.

		NUMBER	MEAN±SD	P value
High Sensitive Troponin-I	Case	20	401.01±625.79	0.01
	Control	20	3.29±1.91	
CK-MB	Case	20	12.19±10.47	0.0001
	Control	20	1.87±0.95	
HOMOCYSTEINE (Positive Cases)	Case	10(50%)	14.97±3.58	0.001
	Control	10	6.12±2.8	
HOMOCYSTEINE (Negative Cases)	Case	10(50%)	5.67±2.55	0.55
	Control	10	6.12±2.8	

REFERENCE

1. Nishtar S.Prevention of coronary heart disease in SouthAsia.Lancet.2002; 360; 1015-8.|2. Sukanta Mandal, Jyothi Bikas Saha et al.Prevalence of Ischemic Heart Disease among urban population in siliguri,WestBengal.India J Community medicine.2009;34(1);19-23.|3. Neki.N.S. Hyperhomocytsteinaemia - An Independent Risk factor for Cardio vascular Diseases . JIACM . 2003; 4; 55-60. |4. ReddyKS.Cardiovascular disease in non-Weatern countries.N Engl J Med. 2004;350;2438-40 |5.Chambers,J.C,Obeid.O.A., Refsum.H.,Ueland.P,Hacckett.D,Hooper.J, Turner.R.M.,Thomson.S.G.and Kooner.J.S.Plasma homocysteine concentration and risk of coronary heart disease in U.K. Indian Asian and Europeanmen.Lancet .2000;355;523-527.|6. ZalakMalav Gadani.Homocysteine-A Novel Risk Factor For AtherovascularDisease. Global Journal For ResearchAnalysis.2015;5-8|7.K.Venkateswarlu,A.Padma Vijayasree.Biochemical Study of Cardiac Markers in Acute Myocardial Infarction.Indian Journal Of Applied Research . 2015;402-403.|8.Bhagyamma.S.N.,Sreenivasulu.U.,Mahesh.G. A Study of homocysteine , creatine kinase and lipid levels in young patients with ischaemic heart disease. IJRHS.2015; 80-85. |9.M.Kemp,J.Donovan,H.Higham and J.Hooper.Biochemical Markers Of Myocardial Injury. British Jouranl of Anaesthesia .2004;93(1);63-73|10. McCully K.Vascular pathology of homocysteinemia: implications for the pathogenesis .Am J Pathol. 1969; 56; 111-28 . | 11.Chamber JC . Obeid OA.Refsum H,Velan P,Hackett D Hooper J,Turner R M,Thomson. S.G.Kooner,J.S. Plasma homocysteine concentrations.Lancet. 2000; 355; 523-27.|12.Refsum H, Smith AD,Ueland PM,Nexo E,Clarke R,McPartilin J,et al. Facts and recommendations about total homocysteine determinations:An expert opinion.Clin Chem .2005;50;3-32.|13.Himmatrao SalubaBawaskar, Parag HimmatraoBawaakar, Pramodini Himatrao Bawaskar.Homocysteine : Often Neglected but Common Culprit of Coronary Heart Diseases.Journal of Cardiovasular Disease Research. 2014; 40-43.| 14.Haynes, W.G.Homocysteine and atherosclerosis:potential mechanisms and clinical implications.Proc.R.Coll.physicians Edinb.200.30;114-122.